

Peculiar Liver Changes in Round Heart Disease in Turkeys

F. NEUMANN, Dr.Med.Vet., Ph.D., U. KLOPFER, Dr.Med.Vet.
and T. A. NOBEL, Dr.Med.Vet., D.T.V.M.
Kimvol Veterinary Institute, Bet-Dagan, Israel

M. S. DISON, B.A., B.V.Sc.
District Poultry Laboratory, Hedera

U. BENDHEIM, Dr.Med.Vet.
Yavne Hatchery

Vet. Rec. (1973). 93. 599-601

SUMMARY.—Specific intracytoplasmic liver cell globules were described in round heart disease in turkeys. These globules are histologically similar to those found in alpha-1 antitrypsin deficiency, an inherited condition in man. A hypothesis on the resemblance between these two conditions is raised.

Introduction

ROUND heart disease in turkey was first described in Canada by Magwood and Bray (1962). Later the condition was reported from the United States (Meyhers & Heppner, 1964; Sautten, *et al.*, 1968; Jankus & Good, 1970; Noren, *et al.*, 1971). Laursen-Jones (1968) and Gallagher and Spence (1970) described briefly a similar condition in Great Britain and noticed the constant liver changes associated with an enlarged heart. The latter authors called this condition cardio-hepatic syndrome.

The condition was characterised clinically by marked depression and listlessness. The birds had ruffled feathers and were retarded in growth (Meyhers & Heppner, 1964). According to our experience, death occurred mainly in birds aged two to 10 weeks; the condition was also found in older turkeys, but without mortality. Male turkeys were predominantly affected. Hunsaker (1971) recorded a mortality rate of four to 10 per cent.

The aetiology of round heart disease in turkey is still unknown. Many factors have been considered including genetic predisposition (Meyhers & Heppner, 1964; Hunsaker, 1971), conditions of management and environmental agents (Jankus & Good, 1970). Noren, *et al.* (1971) suggested that the causal agent was a virus, which resembled morphologically the avian leukosis viruses.

1003546372

In the present study attention was given to the character of the liver changes and to a possible explanation concerning the aetiology of this condition.

Materials and Methods

Seventy-six Nicholas turkeys, aged two to 24 weeks, mostly males, with round heart disease, were necropsied and histological examination was performed.

For histological purposes the heart, liver, lungs and occasionally intestinal segments were harvested. The organs were fixed in neutral 10 per cent. formalin and 7μ paraffin sections were cut. The sections were stained by hematoxylin-eosin. Furthermore, the liver sections were stained by: Mallory's phosphotungstic acid hematoxylin (PTAH), periodic acid Schiff reaction (PAS), Hess and Hollander's toluidin blue stain, Steedman's alcian blue method. Frozen sections, 10μ in thickness were stained by Sudan IV.

Unstained DPX mounted deparaffinised liver sections were examined under ultra-violet light, using a HBO-200 mercury vapour bulb as a light source, dark field condenser and UG1, 2 mm. thick primary, Wratten 2A secondary filter system.

Results and Discussion

The macroscopic changes in the birds were identical to those described by the authors referred to above.

The most evident lesions were the enlargement of the heart (Fig. 1) as a result of the dilatation of both ventricles, with the right one more prominent (Fig. 2), in addition the liver was enlarged, with rounded borders (Fig. 1). The parenchyma was hardened and generally of a greyish-brown colour.

Microscopic examinations Microscopic lesions of the heart were generally not observed. In some turkeys, focal infiltration of mononuclear cells in the myocardium was found. The epicardium in some birds showed a fibrinous exudation with mononuclear cell infiltration. The lungs and the intestines were without pathological changes.

Liver The liver changes were of a degenerative type. Vacuolisation of the liver cells was a constant finding and these cells were occasionally filled with fat. Areas of focal necrosis, bile duct hyperplasia and nodular mononuclear cell infiltration were observed in most of the cases. In older birds, a hyperplastic fibrosis of the liver parenchyma was found. These liver changes closely resemble those previously described by Sautter, *et al.* (1968).

The most distinctive liver changes, in our material, were intracytoplasmic globules, which ranged from two to 14μ in diameter and one to three such globules were found in the affected liver cells. The globules showed a characteristic histological picture with various staining methods. They were anhydric, eosinophile, PAS positive (Fig. 3). PTAH stains the spherules selectively a bluish black and the larger globules were surrounded by a clear halo (Fig. 4). With alcian blue and toluidin blue the globules remained unstained. By ultraviolet light examination the spherules showed a blue white autofluorescence (Fig. 5).

These intracytoplasmic globules seem to be similar



FIG. 1.—Five months old male turkey with an enlarged heart and liver.

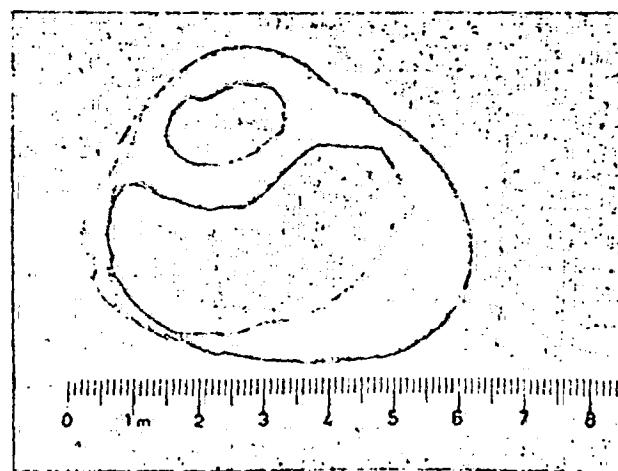


FIG. 2.—Five months old male turkey. Cross section through both ventricles.

to those found by DeLellis, *et al.* (1972), Gordon, *et al.* (1972), Lieberman, *et al.* (1972), Campra, *et al.* (1973) and Glasgow, *et al.* (1973) in alpha-1 antitrypsin deficiency in man, which is considered to be a hereditary condition. This similarity raises the supposition that round heart disease in turkey may be aetiologically related to the above condition in man. Further

1003546373

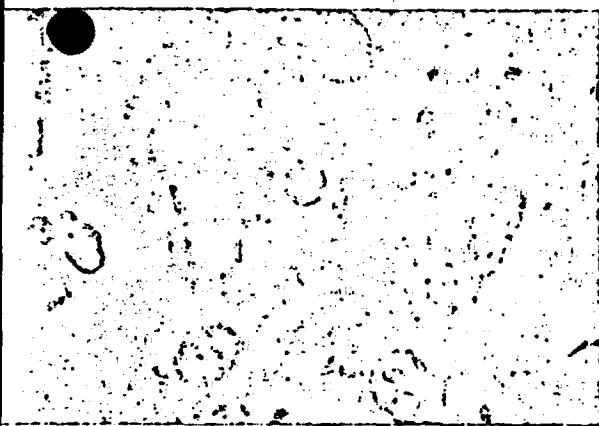


Fig. 3.—Seven weeks old male turkey. PAS positive globules in the cytoplasma of hepatocytes. PAS stain x 630.

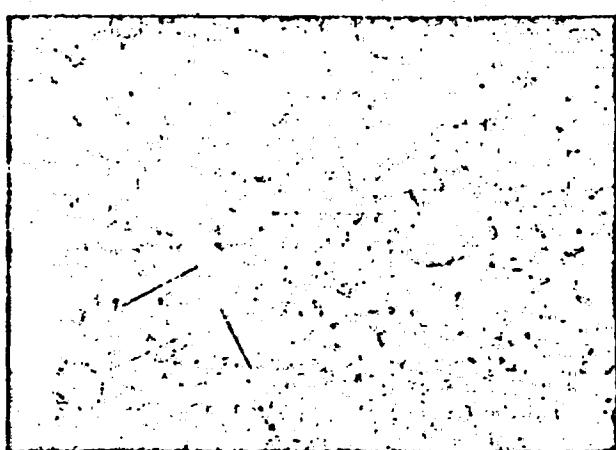


Fig. 5.—Two months old male turkey. Autofluorescence of the intracytoplasmic hepatocytes globules. Unstained section, examined by U.V. light. x 625.



Fig. 4.—Two months old male turkey. Intracytoplasmic hepatocyte globule with a bluish black centre and a clear rim. PTAH stain. x 1250.

support for this hypothesis is the lack of alpha and beta globulins in the blood serum of affected turkeys (Meirom & Trainin). In man the serum alpha globulins are 90 per cent. alpha-1 antitrypsin (Eriksson, 1965).

Acknowledgment.—The authors are indebted to Professor P. P. Levine for the interpretation of some histological sections.

References

CAMPRA, J. L., CRAIG, J. R., PIETERS, R. L., & REYNOLDS, T. B. (1973). *Ann. intern. Med.* 78, 233.
 DeLELLIS, R. A., BALOGH, K., MERK, F. B., & CHIRIFE A. M. (1972). *Arch. Path.* 94, 303.
 ERIKSSON, S. (1965). *Acta med. scandinav.* 117. (Suppl. 432). 1.
 GALLAGHER, J., & SPENCE, J. B. (1970). *Vet. Rec.* 86, 201.
 GLASGOW, J. F. T., LYNCH, M. J., HERCZ, A., LEVISON, H., & SASS-KORTSAK, A. (1973). *Am. J. Med.* 54, 181.
 GORDON, H. W., DIXON, J., ROGERS, J. C., MITTMAN, C., & LIEBERMAN, J. (1972). *Hum. Path.* 3, 361.
 HUNSAKER, W. G. (1971). *Poultry Sci.* 50, 1720.
 JANKUS, E. F., & GOOD, A. L. (1970). *Minnesota vet.* 10, 11.
 LAURSEN-JONES, A. P. (1968). *Vet. Rec.* 83, 156.
 LIEBERMAN, J., MITTMAN, C., & GORDON, H. W. (1972). *Science.* 175, 63.
 MAGWOOD, S. E., & BRAY, D. F. (1962). *Canad. J. comp. Med. vet. Sci.* 26, 268.
 MEIROM, B., & TRAININ, Z. *In preparation.*
 MEYHERS, P., & HEPPNER, R. G. (1964). *Feedstuffs.* 36, 56.
 NOREN, G. R., STALEY, N. A., JANKUS, E. F., & SIEVENSON, J. E. (1971). *Virchow Arch. path. Anat., Abt. A.* 352, 285.
 SAUTTER, J. A., NEWMAN, J. A., KLEVEN, S. H., & LARSEN, C. T. (1968). *Avian Dis.* 12, 614.

Résumé

Des globules spécifiques intracytoplasmiques dans les cellules du foie sont décrits dans la maladie du cœur rond des dindons. Ces globules sont similaires à ceux trouvés dans la déficience d'alpha-1 antitrypsine de l'homme. L'hypothèse de la ressemblance des deux maladies est avancée.

Zusammenfassung

Spezifische kugelförmige intracytoplasmatische Leberzelleinschlüsse werden bei der Rundherzkrankheit von Puten beschrieben. Diese Körperchen ähneln denen die bei Alpha-1 Antitrypsindefizienz des Menschen gefunden wurden. Es wird die Hypothese von der Ähnlichkeit beider Krankheiten aufgeworfen.

1003546374